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NEWS EXPRESS	MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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NEWS LOGIN	Welcome Banner and News Items
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FILE 'HOME' ENTERED AT 12:11:05 ON 24 JUN 2004

=> FIL MEDLINE BIOSIS EMBASE CA SCISEARCH

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 12:11:16 ON 24 JUN 2004

FILE 'BIOSIS' ENTERED AT 12:11:16 ON 24 JUN 2004
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FILE 'EMBASE' ENTERED AT 12:11:16 ON 24 JUN 2004

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FILE 'SCISEARCH' ENTERED AT 12:11:16 ON 24 JUN 2004


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=> s (PCTAIRE protein kinase 1) or ptck1 or crk5 or (pctaire 1)
L1          114 (PCTAIRE PROTEIN KINASE 1) OR PTCK1 OR CRK5 OR (PCTAIRE 1)

=> s antisense or (anti (n) sense) or (complement? (2n)( oligonucl? or nucleo?))
L2       137553 ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMENT? (2N)(OLIGONUCL?
                OR NUCLEO?))

=> s antisense or (anti (n) sense) or (complement? (2n)( oligonucl? or nucle?))
L3       140169 ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMENT? (2N)(OLIGONUCL?
                OR NUCLE?))

=> s l1 and l2
L4             8 L1 AND L2

=> dup rem l8
L8 IS NOT VALID HERE
The L-number entered has not been defined in this session, or it
has been deleted.  To see the L-numbers currently defined in this
session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5             4 DUP REM L4 (4 DUPLICATES REMOVED)

=> d l5 1-4 ibib abs

L5   ANSWER 1 OF 4  CA  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         139:47198  CA
TITLE:                    Use of antisense oligonucleotides to PCTAIRE
                          protein kinase isoenzyme 1 cDNA in treatment of
                          hyperproliferative and neurological diseases
INVENTOR(S):              Freier, Susan M.; Roach, Mark P.
PATENT ASSIGNEE(S):       Isis Pharmaceuticals, Inc., USA
SOURCE:                   PCT Int. Appl., 104 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2003049691      A2   20030619       WO 2002-US39138  20021206
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA,
             UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
             MR, NE, SN, TD, TG
```

US 2003138952 A1 20030724 US 2001-17621 20011207
PRIORITY APPLN. INFO.: US 2001-17621 A 20011207
AB **Antisense** compds., compns. and methods are provided for modulating the expression of PCTAIRE protein kinase isoenzyme 1. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding PCTAIRE protein kinase isoenzyme 1. Methods of using these compds. for modulation of PCTAIRE protein kinase isoenzyme 1 expression and for treatment of diseases are provided.

L5 ANSWER 2 OF 4 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 134:337386 CA
TITLE: Glutamate transporter associated proteins regulating glutamate transport, cytoskeletal organization and chloride flux
INVENTOR(S): Rothstein, Jeffrey D.; Jackson, Mandy; Lin, Glen; Law, Robert; Orlov, Irina
PATENT ASSIGNEE(S): Johns Hopkins University School of Medicine, USA
SOURCE: PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030968	A2	20010503	WO 2000-US29431	20001023
WO 2001030968	A3	20020808		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001011037	A5	20010508	AU 2001-11037	20001023

PRIORITY APPLN. INFO.:
US 1999-161007P P 19991023
US 2000-206157P P 20000522
WO 2000-US29431 W 20001023

AB Glutamate transporter-associated proteins (GTRAP) and cDNAs encoding them are provided. Also provided is a method for identifying a compound that modulates a cellular response mediated by a GTRAP. A method is further provided for identifying a compound that inhibits an interaction between a GTRAP and a glutamate transporter protein. A method is provided for treating a disorder associated with glutamate transport. The proteins were first identified as interacting with the EAAC1 glutamate transporter in a yeast two-hybrid system. Full-length cDNAs encoding three different GTRAPs (3-18, 4-41, and 4-48) were obtained. Each of these proteins bound a different glutamate transporter and the transporter-binding domains are identified. Tissue distribution of the GTRAPs mirrored the pattern of distribution of their cognate transporters. GTRAP3-18 was distributed in a number of major organs while 4-41 and 4-48 were localized to the brain. **Antisense** DNAs from the GTRAP3-18 blocked synthesis of the protein in rats. Synthesis of GTRAP3-18 was also inhibited by retinoic acid.

L5 ANSWER 3 OF 4 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 134:339179 CA
TITLE: Nucleic acids and proteins associated with cancer as antitumor targets
INVENTOR(S): Burmer, Glenna C.; Brown, Joseph P.; Pritchard, David

PATENT ASSIGNEE(S): Lifespan Biosciences, Inc., USA
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030964	A2	20010503	WO 2000-US29126	20001020
WO 2001030964	A3	20010809		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001013397	A5	20010508	AU 2001-13397		20001020
PRIORITY APPLN. INFO.:			US 1999-161232P	P	19991022
			US 2000-693783	A	20001019
			WO 2000-US29126	W	20001020

AB This invention relates to the discovery of nucleic acids associated with cell proliferation, neoplasia, cell transformation, malignant tumor formation and metastasis and uses therefor. The present invention provides a method for cancer diagnosing by detecting the overexpression or the underexpression of a cancer-associated mRNA in the tissue of interest, preferably in liver, breast, prostate, kidney and colon. In another aspect, the invention provides methods for arresting cancer and a method for identifying a modulators of cancer development.

L5 ANSWER 4 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001142585 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11085876
TITLE: Multiple subcellular localizations of **PCTAIRE-1** in brain.
AUTHOR: Le Bouffant F; Le Minter P; Traiffort E; Ruat M; Sladeczek F
CORPORATE SOURCE: CNRS UPR 2212, Institut Alfred Fessard, Gif-sur-Yvette, France.
SOURCE: Molecular and cellular neurosciences, (2000 Oct) 16 (4) 388-95.
Journal code: 9100095. ISSN: 1044-7431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20020420
Entered Medline: 20010308

AB We developed a selective antibody to a synthetic peptide corresponding to an N-terminal sequence of the **PCTAIRE-1** protein. In rodent brain extracts it recognized only the protein doublet characteristic of **PCTAIRE-1**, and this signal is completely abolished by preincubation of the antibody with the immunopeptide. Immunolabeling experiments done with this **PCTAIRE-1**-specific antibody reveal that the protein is widely distributed in the rodent brain as are the mRNAs visualized using an **antisense** riboprobe corresponding to the entire **PCTAIRE-**

1 open reading frame. Two types of **PCTAIRE-1** protein localizations were observed: first a diffuse labeling of almost all brain regions, particularly intense in the molecular layer of the cerebellum and the mossy fiber region of the hippocampus, and second a spot-like localization in the nuclei of large neurons such as cerebellar Purkinje cells and pyramidal cells of the hippocampus. Colocalization with the B23 protein allows one to identify these compartments as nucleoli. Our results suggest a nucleolar function of **PCTAIRE-1** in large neurons and a role in regions containing important granule cell projections.

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NEWS	6	May 12	Polymer links for the POLYLINK command completed in REGISTRY
NEWS	7	May 17	FRFULL now available on STN
NEWS	8	May 27	New UPM (Update Code Maximum) field for more efficient patent SDIs in CAplus
NEWS	9	May 27	CAplus super roles and document types searchable in REGISTRY
NEWS	10	May 27	Explore APOLLIT with free connect time in June 2004
NEWS	11	Jun 22	STN Patent Forums to be held July 19-22, 2004

NEWS EXPRESS	MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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=> FIL MEDLINE BIOSIS EMBASE CA SCISEARCH

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FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 14:26:58 ON 24 JUN 2004

FILE 'BIOSIS' ENTERED AT 14:26:58 ON 24 JUN 2004

FILE 'EMBASE' ENTERED AT 14:26:58 ON 24 JUN 2004

FILE 'CA' ENTERED AT 14:26:58 ON 24 JUN 2004
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FILE 'SCISEARCH' ENTERED AT 14:26:58 ON 24 JUN 2004

=> s (pctaire protein kinase 1) or ptck1 or crk5 or (pctaire 1)
L1 114 (PCTAIRE PROTEIN KINASE 1) OR PTCK1 OR CRK5 OR (PCTAIRE 1)

=> s l1 and inhib?
L2 22 L1 AND INHIB?

=> s l1 (S) inhib?
L3 5 L1 (S) INHIB?

=> dup rem l2
PROCESSING COMPLETED FOR L2
L4 17 DUP REM L2 (5 DUPLICATES REMOVED)

=> s 1-17 ibib abs
L5 0 1-17 IBIB ABS

=> d 1-17 ibib abs
L5 HAS NO ANSWERS
'1-17 ' IS NOT A VALID SEARCH STATUS KEYWORD
Search status keywords:
NONE ---- Display only the number of postings.
STATUS -- Display statistics of the search.
ENTER SEARCH STATUS OPTION (NONE), STATUS, OR ?:end

=> d 1-17 ibib abs l4

L4 ANSWER 1 OF 17 CA
ACCESSION NUMBER: 140:332711 CA
TITLE: Effect of human chorionic gonadotropin in the gene expression profile of MCF-7 cells
AUTHOR(S): Guo, Shanchun; Russo, Irma H.; Lareef, M. Hasan; Russo, Jose
CORPORATE SOURCE: Breast Cancer Research Laboratory, Fox Chase Cancer Center, Philadelphia, PA, 19111, USA
SOURCE: International Journal of Oncology (2004), 24(2), 399-407
CODEN: IJONES; ISSN: 1019-6439
PUBLISHER: International Journal of Oncology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The preventive effect of human chorionic gonadotropin (hCG)-induced differentiation on exptl. mammary carcinogenesis has been reported to be due to the **inhibition** of cell proliferation, increased DNA repair capabilities of the mammary epithelium, decreased binding of the carcinogen to the DNA and activation of programmed cell death genes leading to apoptosis. To further our understanding of the mol. pathway of the hCG action on mammary epithelial cells we have analyzed gene expression profiles of MCF-7 cells treated with hCG for 24, 48, and 96 h, using a DNA microarray consisting of 1176 genes. Comparison of expression between the treated and not treated cells enabled us to identify 48 genes that are affected by this hormone. Importantly, there is a cluster of genes that are overexpressed during the first 24 h and level off thereafter, whereas other genes are maximally expressed at 96 h of

treatment. The results obtained in this study demonstrated that genes regulating cell proliferation, apoptosis, cell trafficking, and DNA repair are significantly affected by hCG in human breast cancer cells in vitro.

REFERENCE COUNT: 113 THERE ARE 113 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 139:363045 CA
TITLE: Genes expressed in atherosclerotic tissue and their use in diagnosis and pharmacogenetics
INVENTOR(S): Nevins, Joseph; West, Mike; Goldschmidt, Pascal
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: PCT Int. Appl., 408 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003091391	A2	20031106	WO 2002-US38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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WO 2003091391	A2	20031106	WO 2002-XA38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003091391	A2	20031106	WO 2002-XB38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003224383	A1	20031204	US 2002-291885	20021112

PRIORITY APPLN. INFO.:
US 2002-374547P P 20020423
US 2002-420784P P 20021024
US 2002-421043P P 20021025
US 2002-424680P P 20021108
WO 2002-US38221 A 20021112

AB Genes whose expression is correlated with an determinant of an atherosclerotic phenotype are provided. Also provided are methods of

using the subject atherosclerotic determinant genes in diagnosis and treatment methods, as well as drug screening methods. In addition, reagents and kits thereof that find use in practicing the subject methods are provided. Also provided are methods of determining whether a gene is correlated
with a disease phenotype, where correlation is determined using a Bayesian anal.

L4 ANSWER 3 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 138:380512 CA
TITLE: Systems and methods for characterizing a biological condition or agent using calibrated gene expression profiles
INVENTOR(S): Bevilacqua, Michael; Cheronis, John C.; Tryon, Victor
PATENT ASSIGNEE(S): Source Precision Medicine, Inc., USA
SOURCE: PCT Int. Appl., 156 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003040404	A1	20030515	WO 2002-US36084	20021108
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003219771	A1	20031127	US 2002-291856	20021108

PRIORITY APPLN. INFO.:
US 2001-348213P P 20011109
US 2001-340881P P 20011207
US 2002-369633P P 20020403
US 2002-376997P P 20020430

AB A method provides an index that is indicative of the state of a subject, as to a biol. condition, based on a sample from the subject. An embodiment of this method includes: deriving from the sample a profile data set, the profile data set including a plurality of members, each member being a quant. measure of the amount of a distinct RNA or protein constituent in a panel of constituents selected so that measurement of the constituents enables evaluation of the biol. condition; and in deriving the profile data set, achieving such measure for each constituent under measurement conditions that are substantially repeatable; and applying values from the profile data set to an index function that provides a mapping from an instance of a profile data set into a single-valued measure of biol. condition, so as to produce an index pertinent to the biol. condition of the subject. The index was determined with resp. to a relevant population which has in common property that is at least one of age group, gender, ethnicity, geog. location, diet,medical disorder, clin. indicator, medication, phys. activity, body mass, and environmental exposure. The biol. conditions include inflammation, diabetes, prostate health or disease, manifested skin, liver metabolism and disease, vascular disease, abnormal cell development, cancer and infectious disease. The method can be used for evaluating the effect on a biol. condition by drugs.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 138:148639 CA
TITLE: Comparison of protein or gene expression patterns of blood cells obtained by microarray to injury database to assess injury
INVENTOR(S): Sharp, Frank R.; Tang, Yang; Lu, Aigang
PATENT ASSIGNEE(S): University of Cincinnati, USA
SOURCE: PCT Int. Appl., 126 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003008647	A2	20030130	WO 2001-US44278	20011128
WO 2003008647	A3	20040325		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003104393	A1	20030605	US 2001-996275	20011128
EP 1425412	A2	20040609	EP 2001-988189	20011128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2000-253568P P 20001128
WO 2001-US44278 W 20011128

AB Methods of injury assessment in an individual include the steps of determining a pattern of expression exhibited by blood cells obtained from an individual and comparing the pattern of expression exhibited by the obtained blood cells to an injury database to assess the injury. The injury database includes genomic injury databases, proteomic injury databases, organ specific injury database, disease specific injury database. The patterns of gene or protein expression are obtained by microarray and analyzed by statistical anal., class prediction, clustering, and computer programs. The genes in the pattern of gene expression comprise acidosis-induced genes, hypoxia-induced genes, glucose-induced genes, ischemia-induced genes. The invention relates to sequences of two human genes which are expressed more highly in Parkinson's individuals. The invention also relates to genes associated with status epilepticus, hypoglycemia, ischemic stroke and hemorrhagic stroke in rat model. The invention also relates to gene expression pattern in males and females, resp. The invention also relates to assessing Parkinson's disease, stroke profusion, drug, neurofibromatosis, manic bipolar depression, migraine headache, schizophrenia, and Tourettes disease based on pattern of expression.

L4 ANSWER 5 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 139:394734 CA
TITLE: Analysis of protein induction in the CNS of SJL mice with experimental allergic encephalomyelitis by proteomic screening and immunohistochemistry
AUTHOR(S): Duzhak, T.; Emerson, M. R.; Chakrabarty, A.; Alterman, M. A.; Levine, S. M.
CORPORATE SOURCE: Biochemical Research Service Laboratory, University of

Kansas, Lawrence, KS, 66045, USA
SOURCE: Cellular and Molecular Biology (Paris, France, Print) (2003), 49(5), 723-732
CODEN: CMOBEF; ISSN: 0145-5680
PUBLISHER: CMB Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Exptl. allergic encephalomyelitis (EAE) is an autoimmune disease characterized by demyelination and inflammatory infiltrates in the CNS, and it is an animal model of multiple sclerosis. Piperonyl butoxide (PBO) suppresses disease in EAE mice, and it exhibits a dual effect on cytochrome P450s that manifests in a transient **inhibitory** phase followed by induction. In order to identify the expression of proteins associated with EAE, a proteomic screening was performed on hindbrain microsomes from control + vehicle, control + PBO, EAE + vehicle, and EAE + PBO female mice. Glucose regulated protein 94 (Grp94) and coagulation factor VIII were among the proteins identified in EAE + vehicle and EAE + PBO mice. Immunohistochem. staining of Grp94 was present in some neurons and oligodendrocytes in hindbrain sections from control animals, and in some cells within inflammatory infiltrates in EAE animals. Since Grp94 (also known as Gp96) can partake in antigen presentation and induction of proinflammatory cytokine expression, its presence in these cells suggests that it may play a role in the pathogenesis of EAE. Coagulation factor VIII is carried and protected by von Willebrand factor. Immunohistochem. staining of von Willebrand factor revealed its presence in some vessels within hindbrain sections from control animals. In EAE animals, the number of labeled vessels was significantly increased, and extracellular granular deposits were observed around labeled vessels indicating that the breakdown of the blood-brain barrier that occurs in EAE permitted its extravasation into the CNS. Addnl. proteins were identified in the different groups of mice by proteomic screening, but confirmation of their expression profile awaits investigations by independent measures.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 138:383243 CA
TITLE: Effects of A1 adenosine receptor overexpression on normoxic and post-ischemic gene expression
AUTHOR(S): Ashton, Kevin J.; Holmgren, Kirsty; Peart, Jason; Lankford, Amy R.; Paul Matherne, G.; Grimmond, Sean; Headrick, John P.
CORPORATE SOURCE: Heart Foundation Research Centre, Griffith University Gold Coast Campus, Southport, 4217, Australia
SOURCE: Cardiovascular Research (2003), 57(3), 715-726
CODEN: CVREAU; ISSN: 0008-6363
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Objectives: To identify potential mol. genetic determinants of cardiovascular ischemic tolerance in wild-type and transgenic hearts overexpressing A1 adenosine receptors (A1ARs). Methods: cDNA microarrays were used to explore expression of 1824 genes in wild-type hearts and ischemia-tolerant mouse hearts overexpressing A1ARs. Results: Overexpression of A1ARs reduced post-ischemic contractile dysfunction, limited arrhythmogenesis, and reduced necrosis by .apprx.80% in hearts subjected to 30 min global ischemia 60 min reperfusion. Cardioprotection was abrogated by acute A1AR antagonism, and only a small number (19) of genes were modified by A1AR overexpression in normoxic hearts. Ischemia-reperfusion significantly altered expression of 75 genes in wild-type hearts (14 induced, 61 down-regulated), including genes for metabolic enzymes, structural/motility proteins, cell signaling proteins, defense/growth proteins, and regulators of transcription and translation.

A1AR overexpression reversed the majority of gene down-regulation whereas gene induction was generally unaltered. Addnl., genes involved in cell defense, signaling and gene expression were selectively modified by ischemia in transgenic hearts (33 induced, 10 down-regulated), possibly contributing to the protected phenotype. Real-time PCR verified changes in nine selected genes, revealing concordance with array data. Transcription of the A1AR gene was also modestly reduced post-ischemia, consistent with impaired functional sensitivity to A1AR stimulation. Conclusions: Data are presented regarding the early post-ischemic gene profile of intact heart. Reduced A1AR transcription is observed which may contribute to poor outcome from ischemia. A1AR overexpression selectively modifies post-ischemic gene expression, potentially contributing to ischemic-tolerance.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 137:180730 CA
TITLE: Human cDNA/DNA molecules and proteins encoded by them with enhanced expression in apoptosis-resistant cell clones, and use thereof in diagnosis, therapeutics and drug screening
INVENTOR(S): Ullrich, Axel; Abraham, Reimar
PATENT ASSIGNEE(S): Max-Planck-Gesellschaft zur Foerderung der Wissenschaften e.V., Germany
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002063037	A2	20020815	WO 2002-EP1073	20020201
WO 2002063037	A3	20031002		
WO 2002063037	C2	20040219		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1364066	A2	20031126	EP 2002-718083	20020201
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004517638	T2	20040617	JP 2002-562773	20020201
US 2004110177	A1	20040610	US 2003-470845	20030731

PRIORITY APPLN. INFO.: US 2001-265631P P 20010202
WO 2002-EP1073 W 20020201

AB The present invention relates to a method for identifying nucleic acid mols. functionally associated with a desired phenotype, such as cancer cell properties, including anti-apoptosis. The method, which allows for generation of expression profiles of genes associated with said desired phenotype, involves a mutagenesis and/or genome rearrangement step, followed by selection of cell clones displaying the desired phenotype. The invention also relates that the method involves the use of the following techniques: fluorescence-activated cell sorting (FACS); nucleic acid microarray (cDNA, genomic or oligonucleotide); protein array;

two-dimensional gel electrophoresis; and/or mass spectrometry. The invention further relates that the disclosed method was used to identify genes, which are differentially expressed in apoptosis-sensitive and apoptosis-resistant cells. Specifically, the invention relates that apoptosis was induced in human cervix carcinoma cell line HeLa S3 by Fas activation. After the selection procedure, only a low number of living cells were present, which had a higher resistance against apoptosis than the parental cell line. MRNA was isolated from these surviving clones, and from the parental cell line, and transcribed into cDNA. CDNA microarray technol. was used to identify about 150-200 genes (cDNA/DNA mols.) that exhibited enhanced expression in apoptosis-resistant clones. The GenBank accession nos. of some of these cDNA/DNA mols. are provided, along with the products encoded by said mols. Still further, the invention relates that most of the apoptosis-associated genes encode protein phosphatases, and kinases. Finally, the invention relates that said nucleic acid mols., and proteins encoded by mols., can be used as targets in diagnosis, therapeutics and drug screening, particularly for disorders associated with dysfunction of apoptotic processes, such as tumors.

L4 ANSWER 8 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 138:20443 CA
TITLE: Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
INVENTOR(S): Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002355079	A2	20021210	JP 2002-69354	20020313

PRIORITY APPLN. INFO.: JP 2001-73183 A 20010314
JP 2001-74993 A 20010315
JP 2001-102519 A 20010330

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises preparing a nucleic acid sample containing mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample containing the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-β estradiol (E2), were found in mice by DNA chip anal.

L4 ANSWER 9 OF 17 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002476045 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12154078
TITLE: Regulation of the CDK-related protein kinase **PCTAIRE-1** and its possible role in neurite outgrowth in Neuro-2A cells.
AUTHOR: Graeser Ralph; Gannon Julian; Poon Randy Y C; Dubois Thierry; Aitken Alastair; Hunt Tim
CORPORATE SOURCE: ICRF Clare Hall Laboratories, South Mimms, Herts EN6 3LD,

UK.. r.graeser@proqinase.com
SOURCE: Journal of cell science, (2002 Sep 1) 115 (Pt 17) 3479-90.
Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20020920
Last Updated on STN: 20030417
Entered Medline: 20030416

AB **PCTAIRE-1** is a CDK-related protein kinase found in terminally differentiated cells in brain and testis, and in many immortalised and transformed cell lines. Bacterially expressed PCTAIRE is completely inactive as a protein kinase, but is a very good substrate for protein kinase A (PKA), which phosphorylates a total of four sites in the N-terminus of **PCTAIRE-1**. Phosphorylation of one of these sites, Ser119, generates a 14-3-3 binding site, which is functional in vitro as well as in vivo. Mutation of another PKA site, Ser153, to an alanine residue generated an activated kinase in transfected mammalian cells. This activity was comparable to that of CDK5 activated by a bacterially expressed, truncated version of p35(nck), p21. Gel filtration analysis of a brain extract suggested that monomeric **PCTAIRE-1** was the active species, implying that **PCTAIRE-1** may not be a true CDK, in that it does not require a partner (cyclin-like) subunit for kinase activity. Finally, we found that various forms of **PCTAIRE-1** transfected into neuroblastoma cell lines could either promote or **inhibit** neurite outgrowth, suggesting a potential role for the **PCTAIRE-1** gene product in the control of neurite outgrowth.

L4 ANSWER 10 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 137:77764 CA
TITLE: Gene expression in bronchoalveolar lavage cells from scleroderma patients
AUTHOR(S): Luzina, Irina G.; Atamas, Sergei P.; Wise, Robert; Wigley, Fredrick M.; Xiao, Hui Qing; White, Barbara
CORPORATE SOURCE: Research Service, Veterans Affairs Maryland Health Care System and Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, USA
SOURCE: American Journal of Respiratory Cell and Molecular Biology (2002), 26(5), 549-557
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Thoracic Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The hypothesis of this study is that activation of cell-mediated immunity with associated macrophage activation occurs in the lungs of scleroderma patients with lung inflammation. Gene expression profiles were determined in bronchoalveolar lavage (BAL) cells from scleroderma patients with and without lung inflammation and control subjects, using DNA array technol. ELISA was used to measure proteins in BAL fluids. Gene expression profiles were similar in BAL cells from patients without lung inflammation and control subjects. Gene expression profiles in patients with lung inflammation showed increased expression of chemokines and chemokine receptor genes, which would lead to migration of T cells, especially type 2 T cells, and phagocytic cells. Protein levels of pulmonary and activated-response chemokine and monocyte chemoattractant protein-1 were elevated. Other changes in gene expression suggested alterations in gene transcription, cell cycle control, vesicle transport, antigen-presenting function, and intracellular signaling. Two anti-inflammatory cytokines, interleukin-1 receptor antagonist and transforming growth factor-β1, had increased expression, consistent with other human fibrotic lung

diseases and animal models of lung fibrosis. These findings suggest recruitment of T cells and chronic macrophage activation in scleroderma patients at greater risk for lung fibrosis, but differ from typical delayed-type hypersensitivity responses, without prominence of type 1 T cells and inflammatory cytokines.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 11 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 135:15154 CA
TITLE: Single nucleotide polymorphisms in coding regions of human genes and primers/probes and methods for detection thereof
INVENTOR(S): Cargill, Michele; Ireland, James S.; Lander, Eric S.
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, USA
SOURCE: PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001038576	A2	20010531	WO 2000-US31639	20001117
WO 2001038576	A3	20020711		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-167334P P 19991124

AB The invention provides nucleic acid segments of the human genome, particularly nucleic acid segments from the coding region of a gene, including polymorphic sites. Allele-specific primers and probes hybridizing to regions flanking or containing these sites are also provided. The nucleic acids, primers and probes are used in applications such as phenotype correlations, forensics, paternity testing, medicine and genetic anal. Thus, total 588 SNPs were identified in 212 genes relevant to cancer, inflammation, heart diseases, cardiovascular diseases and microorganisms by sequencing of target sequences from individuals of diverse ethnic and geog. backgrounds by hybridization to probes immobilized to microfabricated arrays.

L4 ANSWER 12 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 134:337386 CA
TITLE: Glutamate transporter associated proteins regulating glutamate transport, cytoskeletal organization and chloride flux
INVENTOR(S): Rothstein, Jeffrey D.; Jackson, Mandy; Lin, Glen; Law, Robert; Orlov, Irina
PATENT ASSIGNEE(S): Johns Hopkins University School of Medicine, USA
SOURCE: PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030968	A2	20010503	WO 2000-US29431	20001023
WO 2001030968	A3	20020808		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001011037	A5	20010508	AU 2001-11037	20001023

PRIORITY APPLN. INFO.:

US 1999-161007P	P	19991023
US 2000-206157P	P	20000522
WO 2000-US29431	W	20001023

AB Glutamate transporter-associated proteins (GTRAP) and cDNAs encoding them are provided. Also provided is a method for identifying a compound that modulates a cellular response mediated by a GTRAP. A method is further provided for identifying a compound that **inhibits** an interaction between a GTRAP and a glutamate transporter protein. A method is provided for treating a disorder associated with glutamate transport. The proteins were first identified as interacting with the EAAC1 glutamate transporter in a yeast two-hybrid system. Full-length cDNAs encoding three different GTRAPs (3-18, 4-41, and 4-48) were obtained. Each of these proteins bound a different glutamate transporter and the transporter-binding domains are identified. Tissue distribution of the GTRAPs mirrored the pattern of distribution of their cognate transporters. GTRAP3-18 was distributed in a number of major organs while 4-41 and 4-48 were localized to the brain. Antisense DNAs from the GTRAP3-18 blocked synthesis of the protein in rats. Synthesis of GTRAP3-18 was also **inhibited** by retinoic acid.

L4 ANSWER 13 OF 17 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	134:339179 CA
TITLE:	Nucleic acids and proteins associated with cancer as antitumor targets
INVENTOR(S):	Burmer, Glenna C.; Brown, Joseph P.; Pritchard, David
PATENT ASSIGNEE(S):	Lifespan Biosciences, Inc., USA
SOURCE:	PCT Int. Appl., 98 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030964	A2	20010503	WO 2000-US29126	20001020
WO 2001030964	A3	20010809		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001013397	A5	20010508	AU 2001-13397	20001020

PRIORITY APPLN. INFO.:

US 1999-161232P	P	19991022
US 2000-693783	A	20001019

WO 2000-US29126 W 20001020

AB This invention relates to the discovery of nucleic acids associated with cell proliferation, neoplasia, cell transformation, malignant tumor formation and metastasis and uses therefor. The present invention provides a method for cancer diagnosing by detecting the overexpression or the underexpression of a cancer-associated mRNA in the tissue of interest, preferably in liver, breast, prostate, kidney and colon. In another aspect, the invention provides methods for arresting cancer and a method for identifying a modulators of cancer development.

L4 ANSWER 14 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 136:319709 CA
TITLE: Transcriptional profiling reveals global defects in energy metabolism, lipoprotein, and bile acid synthesis and transport with reversal by leptin treatment in Ob/ob mouse liver
AUTHOR(S): Liang, Chien-Ping; Tall, Alan R.
CORPORATE SOURCE: Division of Molecular Medicine, Department of Medicine, Columbia University, New York, NY, 10032, USA
SOURCE: Journal of Biological Chemistry (2001), 276(52), 49066-49076
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Leptin, a hormone secreted by adipose tissue, has been shown to have a major influence on hepatic lipid and lipoprotein metabolism To characterize changes in lipid and lipoprotein gene expression in mouse liver, suppression subtractive hybridization and cDNA microarray anal. were used to identify mRNAs differentially expressed after leptin treatment of ob/ob mice. Ob/ob mice showed a profound decrease in mRNAs encoding genes controlling bile acid synthesis and transport as well as a variety of apolipoprotein genes and hepatic lipase with reversal upon leptin administration, suggesting that leptin coordinately regulates high d. lipoprotein and bile salt metabolism Leptin administration also resulted in decreased expression of genes involved in fatty acid and cholesterol synthesis, glycolysis, gluconeogenesis, and urea synthesis, and increased expression of genes mediating fatty acid oxidation, ATP synthesis, and oxidant defenses. The changes in mRNA expression are consistent with a switch in energy metabolism from glucose utilization and fatty acid synthesis to fatty acid oxidation and increased respiration. The latter changes may produce oxidant stress, explaining the unexpected finding that leptin induces a battery of genes involved in antioxidant defenses. Expression cluster anal. revealed responses of several sets of genes that were kinetically linked. Thus, the mRNA levels of genes involved in fatty acid and cholesterol synthesis are rapidly (<1 h) repressed by leptin administration, in association with an acute decrease in plasma insulin levels and decreased sterol regulator element-binding protein-1 expression. In contrast, genes participating in fatty acid oxidation and ketogenesis were induced more slowly (24 h), following an increase in expression of their common regulatory factor, peroxisome proliferator-activated receptor α. However, the regulation of genes involved in high d. lipoprotein and bile salt metabolism shows complex kinetics and is likely to be mediated by novel transcription factors.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 15 OF 17 CA COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 134:173245 CA
TITLE: Gene expression profile of the aging process in rat liver: normalizing effects of growth hormone

replacement

AUTHOR(S): Tollet-Egnell, Petra; Flores-Morales, Amilcar; Stahlberg, Nina; Malek, Renae L.; Lee, Norman; Norstedt, Gunnar

CORPORATE SOURCE: Department of Molecular Medicine Karolinska Institutet, Karolinska Hospital, Stockholm, 171 76, Swed.

SOURCE: Molecular Endocrinology (2001), 15(2), 308-318
CODEN: MOENEN; ISSN: 0888-8809

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The mechanisms that control life span and age-related phenotypes are not well understood. It has been suggested that aging or at least some of its symptoms are related to a physiol. decline in GH levels with age. To test this hypothesis, and to improve the authors' understanding of the cellular and mol. mechanisms behind the aging process, the authors have analyzed age-induced changes in gene expression patterns through the application of DNA chip technol. In the present study, the aging process was analyzed in rat liver in the presence or absence of GH replacement. Out of 3000 genes printed on the microarrays, approx. 1000 were detected in the rat liver. Among these, 47 unique transcripts were affected by the aging process in male rat livers. The largest groups of age-regulated transcripts encoded proteins involved in intermediary metabolism, mitochondrial respiration, and drug metabolism Approx. 40% of the differentially expressed gene products were normalized after GH treatment. The majority of those transcripts have previously not been shown to be under GH control. The list of gene products that showed normalized expression levels in GH-treated old rats may shed further insight on the action and mechanism behind the pos. effects of GH on, for example, fuel metabolism and body composition observed in different animal and human studies.

REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 16 OF 17 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 135:56831 CA

TITLE: Identification of differentially expressed genes in human gliomas by DNA microarray and tissue chip techniques

AUTHOR(S): Sallinen, Satu-Leena; Sallinen, Pauli K.; Haapasalo, Hannu K.; Helin, Heikki J.; Helen, Pauli T.; Schraml, Peter; Kallioniemi, Olli-P.; Kononen, Juha

CORPORATE SOURCE: Department of Pathology, Laboratory of Molecular Pathology, Tampere University Hospital, Tampere, FIN-33521, Finland

SOURCE: Cancer Research (2000), 60(23), 6617-6622
CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB New genomic large-scale screening techniques have made the task of establishing an accurate mol. fingerprint of cancer cells feasible. Here, we have used a two-phase strategy for identification of mol. alterations in gliomas. First, cDNA microarrays (Clontech Labs., Inc., Research Genetics) were used to pinpoint differentially expressed genes between normal brain and diffuse astrocytomas (grades II-IV), and between a primary tumor and a later tumor reoccurrence in the same patient. More than 200 gene expression alterations were detected from glioblastomas, whereas relatively few changes were seen in grade II and grade III tumors. The most distinct progression-related expression change was the up-regulation of the insulin-like growth factor binding protein 2 (IGFBP2) gene. Second, a high-d. tissue microarray of 418 brain tumors was constructed and used for clin. validation of gene expression changes.

Strong expression of IGFBP2 was associated with progression and poor patient survival in diffuse astrocytomas (P < 0.0001). Third, comparisons of the data between (a) multiple spots retrieved from one predefined tumor region (IGFBP2 and vimentin immunohistochem., 20 tumors) or between (b) standard slides and arrayed tissues (p53 immunohistochem., 42 tumors) revealed very little variation. In conclusion, the combined use of DNA microarrays and tissue microarrays offers a powerful strategy for rapid identification and thorough characterization of differentially expressed genes in gliomas.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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TITLE: Identification of tudor repeat associator with PCTAIRE 2 (Trap). A novel protein that interacts with the N-terminal domain of PCTAIRE 2 in rat brain.

AUTHOR: Hirose T.; Kawabuchi M.; Tamaru T.; Okumura N.; Nagai K.; Okada M.

CORPORATE SOURCE: T. Hirose, Division of Protein Metabolism, Institute for Protein Research, Osaka University, 3-2 Yamadaoka, Suita, Osaka 565-0871, Japan. hirose@protein.osaka-u.ac.jp

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SUMMARY LANGUAGE: English

AB PCTAIRE 2 is a Cdc2-related kinase that is predominantly expressed in the terminally differentiated neuron. To elucidate the function of PCTAIRE 2, proteins that associate with PCTAIRE 2 were screened by the yeast two-hybrid system. A positive clone was found to encode a novel protein that could bind to PCTAIRE 2 in vitro as well as in vivo, and was designated as Trap (tudor repeat associator with PCTAIRE 2). The overall structure of Trap shows no significant homology to any proteins, but contains five repeated domains (the tudor-like domain), conserved in Drosophila tudor protein. Trap associates with the N-terminal domain of PCTAIRE 2 through its C-terminal domain, which contains two tudor-like domains. **PCTAIRE 1**, but not PCTAIRE 3, can also associate with Trap. Trap is predominantly expressed in brain and testis, and gradually increases during brain development throughout life, consistent with the expression pattern of PCTAIRE 2. Immunoreactivities for PCTAIRE 2 and Trap were colocalized to the mitochondria in COS 7 cells. Immunohistochemical analyses showed that PCTAIRE 2 and Trap were distributed in the same cell layer of the cerebral cortex and cerebellum. These findings suggest that Trap is a physiological partner of PCTAIRE 2 in terminally differentiated neurons.